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Lymphadenopathy in the Pediatric Patient: A Journey Through a Challenging Diagnosis

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ABSTRACT

This case report describes a 16-year-old male diagnosed with cat scratch disease. Initially assessed by pediatricians for worsening submandibular lymphadenopathy and was advised to undergo lymph node excisional biopsy to exclude malignancy. Cat scratch disease (CSD) is a relatively common zoonotic infection transmitted from a cat scratch or a cat bite to humans. It manifests with acute lymphadenopathy or lymphadenitis, usually affecting cervical, submandibular, or axillary lymph nodes, with or without systemic symptoms. A detailed patient history, especially about animal exposure, is crucial to suspecting the condition, which can be confirmed or ruled out later with simple serological tests. The patient was suspected of having CSD based on a history of a cat bite, received laboratory confirmation of the disease, and was successfully treated with proper antibiotics.

Keywords: *Bartonella henselae*; cat scratch disease; lymphadenitis; lymphadenopathy.

INTRODUCTION

Cat scratch disease is typically a self-limiting infectious disease caused by *B. henselae*. Since cats are the main reservoirs of the bacterium, this zoonotic infection transmits to humans when cat saliva comes in contact with broken skin, mucous membranes (including conjunctiva), cat bites, or scratches.[1] On the other hand, a cat acquires the infection horizontally from another cat via a vector, Ctenocephalides felis, a cat flea.[2]

In most infected children, the infection manifests as a localized skin and lymph node disorder near the site of the microorganism inoculation. Rarely it can disseminate and infect the reticuloendothelial system (liver and speel), eyes, and CNS. Unlike a disseminated infection, a localized infection is generally a self-limiting condition that can cause life-threatening complications.[3]

Regional lymphadenopathy, which usually starts around two weeks after the pathogen is inoculated in the skin and typically involves axillary, cervical, epitrochlear, submandibular, and supraclavicular nodes, is a hallmark of the infection.[4] The size of enlarged nodes ranges from 1 to 5 cm but may enlarge to 8 to 10 cm. Lymphadenopathy associated with CSD usually resolves in one to four months.[5]

CASE

A 16-year-old previously healthy male was admitted to our center on the 10th day of progressively worsening unilateral tender submandibular lymphadenopathy, high-grade fever, dysphagia, difficulty in mastication, and vomiting.

Initially, he developed a lump jaw for which he consulted a pediatrician who ruled out more common causes of acute lymphadenopathy in children and recommended waiting for spontaneous resolution. However, within a few days, the lump rapidly increased in size, interfering with regular oral intake due to pain in chewing and dysphagia. The patient started with cefixime on the fifth day of illness but developed a high-grade fever. The general condition worsened, and he was consulted by a surgeon in an outpatient setting, who recommended performing an excision of the node for further histological assessment to rule out possible malignancy. Finally, the patient was admitted to our center. At the admission, he was febrile, toxic appearing, and mildly dehydrated. CSD was suspected since other common infectious causes of the condition were already ruled out, even though the patient could not recall an actual scratch or bite from his domestic cat.

With ultrasound, a round-oval-shaped lesion 3.3 x 2.9 cm in size was detected in the area of the right lower jaw at a depth of 0.7 cm from the skin. The lesion had smooth contours of mixed echogenicity and echostructure with limited vascularization. Based on this ultrasound finding, it was most consistent with an infected lymph node.

CBC was unremarkable, but CRP level was elevated to 118.9 mg/L (n <5mg/L).

The patient was started with ampicillin-sulbactam intravenously and oral azithromycin, along with other supportive measures.

The submitted serological test for *B. henselae* came back positive, confirming the CSD diagnosis.

After initiating the antibiotics, the patient became afebrile in 3 days; his general condition improved, the mandibular lump started to reduce in size, and its tenderness on palpation decreased despite physical examination findings. The repeated ultrasound demonstrated an increase in the size of the lymph node to 4.6 x 3.0 cm with peripheral hemorrhagic foci. Repeated labs showed unremarkable CBC and the CRP level normalized 5.97mg/L (n < 5mg/L).

Despite the ultrasound finding, the patient's condition was satisfactory, and he was discharged from the hospital with a proper prescription and a follow-up plan..

DISCUSSION

As mentioned, CSD is a relatively common zoonotic infection caused by *B. henselae*, which transmits from cats to humans through saliva.[1] Even though the infection is self-limiting in most cases, the lymphadenopathy may sometimes last up to 4 months.[5] This prolonged course of illness may reclassify acute lymphadenopathy to subacute, which has various infectious and noninfectious causes, including pediatric malignancies. The differential diagnosis of CSD includes conditions like (but not limited to) Mycobacterial infections, bacterial adenitis, associated lymphadenopathy (EBV, CMV, HIV), lymphomas, and sarcomas.[6,7]

Since most patients with worsening unilateral acute lymphadenitis will very likely receive empiric antibiotics, which, according to recommendations, usually cover GAS, *S. aureus,* and oral anaerobes (in those with poor dentition) (amoxicillin-clavulanate), the empiric regimen does not cover *B. henselae* well.[8] Despite the self-limiting nature of the infection, antibiotics (azithromycin) are recommended to prevent dissemination and disease progression.[5,9,10]

As mentioned, in our case, the patient had developed worsening unilateral submandibular lymphadenitis that did not respond to initial treatment with cefixime (no anaerobic and Bartonella coverage). Rapidly increasing size and failure to respond to antibiotics were the most likely reasons to suspect a serious condition, which urged doctors to recommend the excision of the node with further histological evaluation of the sample.

The diagnosis of CSD is based on the characteristic clinical features- regional tender lymphadenopathy with or without preceding skin lesion (at the inoculation area) and a history of recent cat or flea contact.[5]

Serologic tests are ordered to support a presumptive diagnosis of the infection.[1]

CONCLUSIONS

This case report shows that Bartonella infection as a causative agent of lymphadenopathy might be underestimated in some cases. No proper tests might be requested, so that specialists might perform unnecessary investigations and invasive procedures.

Precise medical history taking is required in such cases to suggest a *B. henselae* infection and start proper treatment. Educational activities and the publication of case reports and studies for doctors and residents will increase awareness about the disease and minimize clinical mistakes in the future.

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